

underwent clinical and laboratory evaluations, ECGs, chest X-rays, Holter recordings, echocardiograms, signal averaged ECGs, and invasive electrophysiologic studies.

**Results:** Twelve pts had structurally normal hearts and 2, arrhythmogenic RV dysplasia. VT was induced by exercise in all pts and not inducible with programmed stimulation. Corticosteroid therapy: prednisone, mean dose 28 mg/day, range 25-50 mg. Ten pts had complete disappearance of VT after 1 month of therapy (generally after 10 days). No effect was seen in 4 pts. During the follow-up 4 pts showed reappearance of VT after therapy withdrawal, with abolition of the arrhythmia after a second 2 month trial with 50 mg of prednisone daily.

**Conclusion:** The use of corticosteroids should be considered as an alternative therapy for pts with RVOT-VT. Further follow-up studies are required.

5:15

### 828-6 Right Ventricular Outflow Tract Tachycardia in Women: Premenopausal, Gestational and Menopausal Exacerbation

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**Background:** Right ventricular outflow tract tachycardia (RVOT-VT) is characterized by repetitive bursts and PVCs with a left bundle branch block and inferior axis morphology with VT typically precipitated by exercise.

**Methods:** Sixteen consecutive women (mean age = 46, range 29-64) who presented with RVOT-VT were interviewed to determine if predictable exacerbation in symptomatic VT was associated with known periods of increased hormonal fluctuations. Specifically, the patient was asked whether they experienced any predictable exacerbation of VT, if a positive response was received, the patient was asked whether RVOT-VT occurred with predictability with stress, exercise, or at a time of premenstrual, perimenopausal, or gestational periods. Eight pts were premenopausal and 2 were never pregnant.

**Results:** Reported as the frequency of a distinct, predictable exacerbation in RVOT-VT.

Unpredictable	3/16 (19%)
Stress	4/16 (25%)
Exercise	1/16 (6%)
Premenopausal only	6/16 (38%)
Perimenopausal only	4/3 (44%)
Gestational only	1/14 (7%)
Total with hormonal shifts	11/16 (69%)

**Conclusions:** In women, RVOT-VT appears to be predictably exacerbated by gender specific hormonal fluctuations that occur with menopause, gestation and during the premenstrual period. Precipitation by exercise is uncommon.

### 829 New Serologic Markers for Risk Stratification in Ischemic Syndromes

Monday, March 30, 1998, 4:00 p.m.-5:30 p.m.  
Georgia World Congress Center, Room 267W

4:00

### 829-1 "Low-Risk" Chest Pain Patients may be at High Risk for Cardiac Events: Prospective Use of Troponin T for Risk Stratification

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Cardiac troponin T (TnT) has shown prognostic value in chest pain (CP) patients (pts) with ischemic ECG changes. However, in "low risk" CP pts who undergo expedited evaluation in chest pain units, little is known about the diagnostic and prognostic value of TnT.

**Methods:** "Low-risk" CP pts were defined as having <7% MI risk (Goldman et al. NEJM 1988; 318: 797) with no new ST/T changes. We prospectively evaluated 414 pts who met these criteria. CKMB and TnT (ELISA) were drawn simultaneously  $\geq 10$  hrs after CP onset. Positive (+) CKMB was defined as 25 ng/ml, +TnT >0.1 ng/ml and CAD  $\geq 70\%$  stenosis by angiography.

**Results:** TnT was + in 37/414 (9%) pts, of whom 20 (54%) had +CKMB. TnT+ pts (vs TnT- pts) had a higher prevalence of typical CP (46% vs 20%,  $p = 0.001$ ), diabetes (46% vs 24%,  $p = 0.003$ ), old  $\pm$  age (59% vs 51%,  $p = 0.002$ ) but no difference ( $p > 0.05$ ) in gender, hypertension, hypercholesterolemia, smoking or prior MI hx. Of the 32 TnT+ pts who underwent angiography, 90% had significant CAD [ $\geq 70\%$  1 vessel (V), 40% 2V, 23% 3V] with 100% occlusion present in 47%. Follow-up was obtained in 94% of pts (mean 264  $\pm$

129 days). Multiple logistic regression of TnT, CKMB and clinical risk factors revealed that only a +TnT was a significant predictor for the occurrence of subsequent death or MI (6 events) [odds ratio (OR) = 11.0, 95%CI 2.1-56.0,  $p = 0.001$ ] whereas a combined event rate (death, MI, readmission for CHF or unstable angina; 55 events) was significantly associated with both +TnT (OR = 2.7, 95%CI 1.2-6.0,  $p = 0.014$ ) and diabetes (OR = 2.3, 95%CI 1.3-4.2,  $p = 0.007$ ).

**Conclusions:** In a "low-risk" CP population, (1) TnT is elevated almost twice as frequently as CKMB; (2) +TnT is associated with multi-vessel CAD; and (3) +TnT is an independent predictor of long-term adverse cardiovascular events.

4:15

### 829-2 Value of Markers of Myocardial Injury and Intracoronary Thrombus in Risk Stratification of Patients Presenting to an Emergency Department With Chest Pain

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As the pathogenesis of acute coronary syndromes involves thrombus formation, followed by myocyte injury, determining the levels of markers of thrombus formation along with markers of myocyte injury may determine patients (pts) at risk for future cardiac events. We determined the levels of serum troponin I (TnI), myosin light chain 1 (MLC1), myoglobin (Mb) and urine fibrinogen A (FPA) in 245 pts (Mean Age 52, 46% female) presenting to an ED with chest pain. Follow up cardiac events were defined as cardiac death, myocardial infarction (MI), unstable angina (UA) and coronary revascularization occurring during the 6 months following pts' initial presentation. During follow up 68 patients developed new cardiac events; 10 pts died, 13 had MI, 50 had UA, and 28 were revascularized. Using univariate analysis, FPA was the only marker significantly predicting death ( $p = 0.026$ ), MI ( $p = 0.02$ ), UA ( $p = 0.02$ ) as well as combined events ( $p < 0.0001$ ). None of the markers were predictive of long term revascularization. Using logistic multivariate regression analysis, FPA was the strongest predictor of cardiac events ( $p = 0.0002$ , OR = 3.6) followed by TnI ( $p = 0.046$ , OR = 2.0) while Mb and MLC1 were not significant. Urine FPA identifies pts at high risk for future cardiac events. Since FPA is a marker of thrombus, intracoronary thrombus may be a more significant predictor of outcome in an unselected population of chest pain pts.

4:30

### 829-3 Positive Serum Markers in Patients With Chest Pain in the Emergency Department Have Prognostic Significance

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**Background:** Millions of patients present annually with chest pain, but only 10% have myocardial infarction (MI). We recently reported comparative sensitivity and specificity (sens/spec) of available markers for early and late diagnosis of MI. However, optimal interpretation requires long-term prognostic follow-up data.

**Methods:** 30 day and 6 month follow-up of 955 consecutive patients with chest pain were enrolled in a prospective, multicenter, double-blind study. The prognostic significance of MB CK subforms, troponin I and T, myoglobin and Total CK to predict death and revascularization (RV) was ascertained.

**Results:** MI was diagnosed in 119 by CK MB mass and unstable angina (UA) in 203 by clinical criteria. Of patients with UA, CK MB subforms were positive in 29%, myoglobin, 24%, troponin I, 20%; troponin T, 15%; and CK MB mass, 15%. Follow-up at 30 days and 6 months in 624 and 724 patients respectively showed mortalities of 2.8% and 4.14% respectively. Six-month mortality was 5.6% in MI, 4.4% in UA, and 3.0% in other patients ( $p < 0.025$ ). RV occurred in 9.3% at 6 months. A positive test on each of the markers except myoglobin was predictive of RV (relative risk (RR) 1.41 to 2.1,  $p < 0.05$ ). The composite endpoint of death or RV occurred in 107 patients by 6 months and a positive result on each of the markers was predictive of this (RR 1.43-2.0,  $p < 0.05$ ). 82% of patients with MI were positive for CK MB subforms in the Emergency Department (ED) within 1 hour of arrival. The RR of death or RV for non-MI patients who test positive on each of these markers was greater than 1.0 but did not reach statistical significance.

**Conclusions:** Early markers (CK MB subforms and myoglobin) positive in patients with chest pain shortly after arrival in the Emergency Department have diagnostic and prognostic significance as do late markers (troponin T, I and total CK MB) whether in patients with MI or UA.